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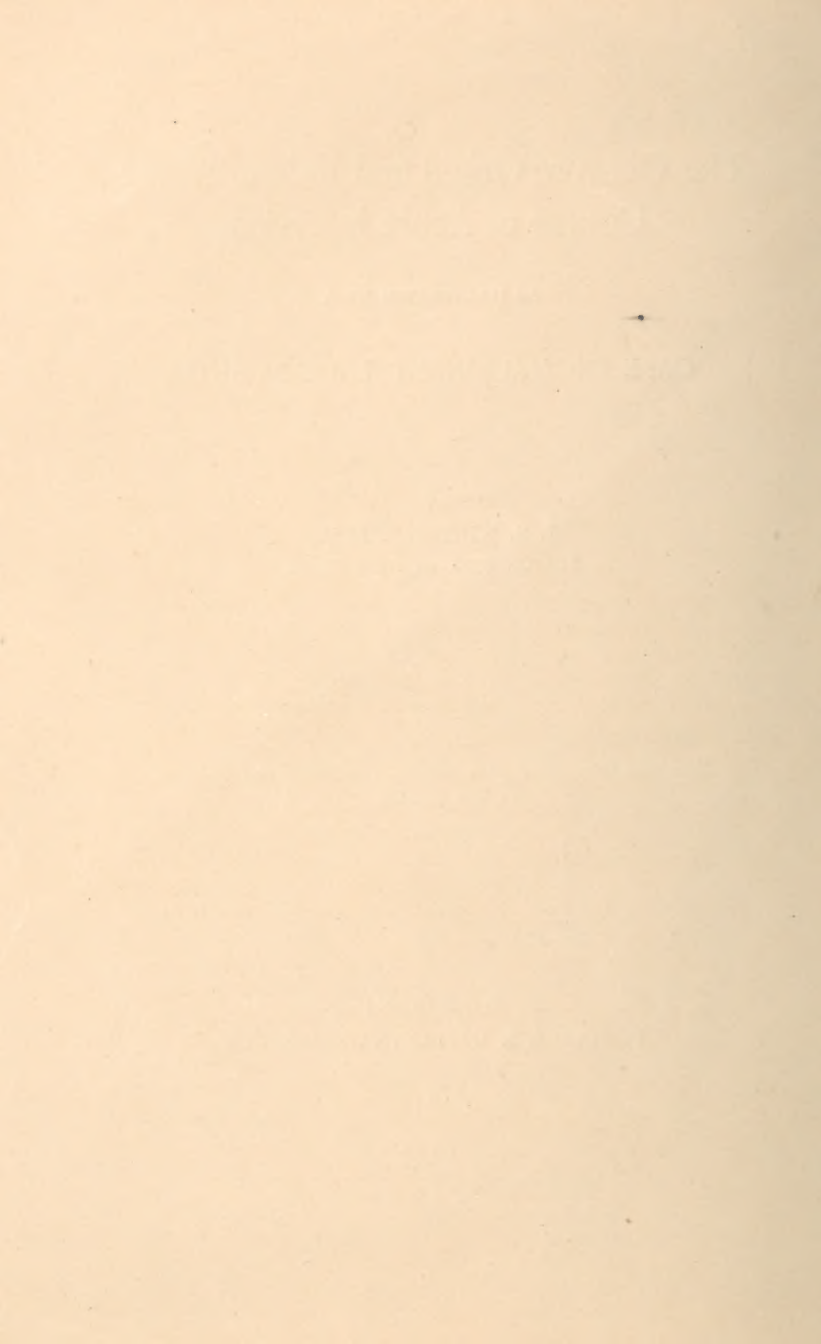
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THE OBSCURE ORIGIN AND INDETERMINATE COURSE OF ACUTE INFECTION, AS ILLUSTRATED BY A CASE OF MALIGNANT ENDOCARDITIS.¹

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IN the near past, when the theory of infection now in vogue was first authoritatively set forth in its full and fascinating proportions, it seemed as if there could no longer arise cases of doubtful nature or uncertain origin. The so-called spontaneous development of infectious maladies in man apart from any ruling epidemic was artificially paralleled by Bouchard when he provoked in healthy animals, without wounding, the rapid appearance of microbes in the blood, by the application of depressing causes such as cold, fright, fatigue.²

By the action of such commonplace influences the pathogenic bacteria which inhabit our cavities, and which ordinarily remain harmless, are enabled to penetrate into and multiply in the fluids. It is thus that infectious diseases become generalized or aggravated in the system, and set up the phenomena of septic fever.

Some of these phenomena are headache, pain in the back and limbs, secretory disturbances, coma, convulsions, delirium; and perhaps there must be added to this already formidable train, paralyses, both of periph-

¹ Read before the Boston Society for Medical Observation, March 5, 1894.

² *Theorie de l'infection, verhandlungen des x internationalen medicin. Congresses*, a. t. 49.



eral nerves and of nerves proceeding from the bulbar centres on the integrity of which the instant life depends.

We are no longer permitted, as in the beatific past, when bacteria were not in sight and ptomaines were not even suspected, to talk of brain fever when the septic process is chiefly determined to the cerebral functions, nor of rheumatic fever when the back and limbs receive the rebound from the shock of poison dealt to the trophic nerve centres. Still traces of what was our state of blissful ignorance remain, and in the light of later knowledge show as black spots on our sunshine. Cases yet arise where a disease ultimately determined to be of infectious origin masks itself behind groups of symptoms capable of being quite otherwise interpreted. The infectious agent, whatever microscopic form it takes, induces a septic fever, which acts with varying intensity and by multi-form combinations on the nervous system and on different organs and functions. Soon or late, the integrity of some important organ may be steadily invaded, or an acute inflammatory process may be localized in some tissue; and whether these complications occur or not, the phenomena of septicæmia, pure and simple, may dominate to the end. Again, the infection rather than the localization may have had the minor rôle, proceeding without the ranges of temperature commonly associated with septic processes. Nervous disturbances apparently or manifestly functional may prevail over either thermic or adynamic, to that degree that when the disease finally becomes localized by signs or symptoms ordinarily unmistakable, these signs and symptoms are misinterpreted because disconnected from their customary relationship.

Material for cultures is not always attainable during the course of the disease; and when a fatal issue has not been averted, and when the pecuniary or vital wants of survivors combine with the scientific interest

of the physician to seek what of verity autopsy may reveal, the early solicitude of the funeral undertaker to preserve the integrity of the mortal remains by the injection of poisonous chemicals will probably have interposed an effectual barrier to the inquiries of the bacteriologist and the microscopist.

A fatal case of disease of infectious origin simulating multiple neuritis, but proven by autopsy to have been acute endocarditis, supplies the clinical material to illustrate the foregoing remarks.

The case was that of a man thirty-four years of age, by occupation a letter-carrier, and thus exposed to the influences of cold and fatigue. His father died of typhoid fever; his mother survives in impaired health, with signs of cardiac trouble. There is no family history of rheumatism. The patient was about five feet four inches in height, spare in flesh, nervous temperament, good habits. At the age of thirteen, after the shock of a fall and exposure to wet, he had a prolonged attack of inflammatory rheumatism with cardiac and pleuritic complications; but recovery, though slow, was complete, and for twenty years thereafter he had no illness requiring medical attention. A less satisfactory state of health dates from about a year ago, when he yielded for part of a week only to an attack of gripal influenza. Following that came annoyances of nasal catarrh and anal fissure, for which troubles he sought relief as a hospital out-patient. These ailments, and a cold pain, as he termed it, above the left hip, accompanied noticeable diminution of strength. Early last November a widowed sister living with him died after a brief illness, leaving two orphan children to his charge. Soon after this event, prodromic symptoms began, and for two months continued to accumulate. These symptoms were lessened appetite; frequent perspirations; pains in shoulders, legs and soles of feet; cold throat; stiff and tired ankles, worse in morning. His urine had been remarked habitually thick.

During the week previous to the final yielding to illness, his nights were marked by sweats, and by cold, restlessly-moving feet; and he remained in bed two days before going again upon his route. In the night of Sunday, January 14th, he aroused the family by a scream of pain, and said it was as if his two feet were cut off. Pain lasted all night, yet he went to work Monday, but ate no dinner. While delivering, pains caught him, first in chest from side to side, passing to back of legs below knees; and he returned home with stooping shoulders and limping gait. Nevertheless, that evening he walked half a mile and back to consult a doctor, and got a tonic. Tuesday, the 16th, he put his last day's work between two restless nights. Wednesday morning, the 17th, at 3 o'clock, he arose and went down stairs, unable to rest from pains in sides and chest, in legs and in feet. That day a physician was summoned. The patient complained of stiff cords around ankles and numbness of soles. Thursday, red streaks were seen on the feet. The hands and arms had become painful, and the abdomen was hard. Friday, the jaws and the back of the neck were involved in the suffering. The history to this point is gathered from patient and his mother.

I first saw him on the evening of Saturday, January 20th, in consultation; and at the joint request of physician and patient, I became associated with the former in the guidance of the case. The patient was lying on his back, with an aspect of helpless unrest, talkative and moaning; his countenance expressive of anxiety. The pulse was 104, fair quality; respiration 26; temperature 101.2° ; tongue clean, but dryish; thirst. He complained of shooting pains down the thighs and legs, of burning tenderness of the outer and inner aspects of thighs, and of numbness of the soles just anterior to the heels — all dating from Wednesday; of pains in the arms (especially about the elbows), with numbness of the two outer fingers of each hand — from

Thursday ; of pain in the lower jaw, below articulation on each side — since Friday : of concurrent pain about the region of the lower ribs on each side, especially the right. On examination, there was no redness nor swelling of joints nor elsewhere. The tenderness elicited by touch was regional, not following the course of large nerves nor in joints ; especially noted at the upper portion of the calves and at the anterior surface of the soles, including toes, especially of right foot. Other points or patches of tenderness to touch were over the ramus of the jaw on each side. Moving or flexing the toes caused pain. There was a degree of rigidity of the lower limbs, and of resistance to passive motion, and such motion caused pain ; but the pain was vaguely localized, and not in joints. Hands and arms showed less rigidity and little tenderness. He moved the jaw with difficulty, complaining of pain below articulations. It was noted that the apex beat of the heart was in the mammary line in the sixth interspace ; but there was no diffused nor heaving impulse, and the heart sounds were clear, unaccompanied by murmur and of normal rhythm, and so remained throughout. The plantar and patellar reflexes were absent. Albumen was absent from the urine, which was heavy with amorphous urates. The patient lived for ten days longer, and died the 30th of January.

During this period of daily observation, there was gradual abatement of numbness, of localized tenderness, and of muscular rigidity ; no restoration of tendon reflexes ; muscular wasting rapid and extreme.

On the 24th, it was noted that pains had appreciably abated within two days, and none remained in sides of chest. Patient could move limbs more easily, and open jaws more readily. He still shrank from touch at the plantar surface of the toes and adjacent part of the soles, and still kept the limbs rather rigid, especially his hands and arms, with fingers spread apart, moving and flexing like one slowly scratching.

On the 25th, he sat on the edge of the bed a few minutes after passing urine.

On the 26th, there were painful and unsuccessful attempts at micturition, a resort to the catheter, and (later) successful voluntary effort. This day tenderness to touch and numbness were all gone. A few petechial spots were seen on the legs. He still kept a certain rigidity of joints on passive movements, as if fearful of hurt, but could relax and move painlessly. The type of fever became more and more distinctly typhoidal, the tongue more dry and furred; no sordes. Delirium of sufferings and of travel, active at first, became somewhat more quiet. He wandered all over the country and encountered all kinds of trouble; he never knew where he was, but was sure he was not at home, though he got almost there; yet he knew every one about him. He had occasional "wild spells," when he wanted to get up and off, but had little strength to exert. There were moments of quick, labored respiration, but not often; scarcely any cough.

His temperature had no regular oscillations, but ranged oftenest from 100.5° to 101.5° ; but the morning of the 23d it reached 103.5° , and the evening of the 24th and the following morning it was 103° . The pulse generally went with the temperature, though not invariably; it varied between 104 and 120, reaching (exceptionally) 130.

The last day I did not see him; but there was choking on attempts to swallow, collapse with cold sweat, and he died with gradual heart-failure.

Dr. Wm. T. Councilman performed an autopsy, Thursday, February 1, 1894: "Anatomical diagnosis, acute endocarditis of aortic and mitral valves. Body of medium size, slightly built, somewhat emaciated. The body had been injected by an undertaker, so that little could be told of the degree of congestion, etc., of internal organs. The peritoneum was smooth, and there were no lesions in any of the abdominal organs.

The spleen was large and rather soft. Both lungs slightly adherent. On section, a slight muco-purulent secretion in some of the smaller bronchi. The heart was of ordinary size, the cavity of the pericardium obliterated by adhesive pericarditis. Myocardium pale and easily torn. The valves of the right heart were normal. On the aortic valves, just along the line of closure, there were numerous projecting granulations. The valve about the seat of these vegetations was thickened. Similar vegetations were along the free edge of the mitral valve, and in one place extended over the auricular surface of the valve up to the auricle. The tissue here was thickened, infiltrated, and small ecchymoses were here and there visible. The nerves of the lower extremities were removed for examination. Owing to the injection of the undertaker, bacterial cultures which would probably have thrown much light on the case could not be made. I think there is little doubt from the character of the lesions in the heart that there has been an infection with either the *diplococcus pneumoniae* or the *streptococcus*."

The result of this autopsy was the revelation of an unsuspected seat and form of localized inflammation. True, the chief signs and symptoms on which a diagnosis of infectious neuritis was founded had gradually abated during the week which followed my introduction to the case. On the other hand, the group of symptoms representing septic infection had kept their unrelenting sway, dominating and supplanting, while no signs were detected nor symptoms intruded to draw attention to a lesion of cardiac valves.

Thus, prior to the autopsy, there still remained the possible alternative that death was wholly due to the effect of the toxins of infection acting on the higher nerve centres. The inference that such was partly the case, that the cardiac lesions were in fact subordinate in determining the manner of death, derives support from the symptom of occasional rapid, labored

breathing, and from the paralysis of muscles of deglutition noted some hours before death.

The temptation is here presented to follow to their natural conclusion these observations on the uncertainty attending the origin and course of acute infection, by some reference to like uncertainties in the fatal ending.

This final point can be illustrated by a case of possible multiple neuritis comparable to the foregoing one, but introduced here only in synopsis. It was the case of a young man of remarkably neurotic constitution by inheritance, whose father and two paternal relatives had died suddenly in acute agony — two of them with precordial pain, the third (a girl of sixteen) of toothache, so-called. An autopsy on the father showed no disease of brain, heart nor kidneys. The son had been pushed to the verge of nervous break-down by overwork and use of tobacco; still further depressed in his vitality by two illnesses supposed to be *la grippe* — one marked by semi-stupor, numbness and bad head, the other by vomiting, headache, pain of back and limbs, and rigidity.

He is out for an evening's enjoyment, perhaps gets chilled, has next morning a convulsive seizure, nearly or quite unconscious, some fever and transient albuminuria; five days of disability with spinal and lumbar pains. Another seizure; nine more days of suffering, involving legs as well as back. Another seizure; pains more torturing, and involving arms and somewhat chest, as well as back and legs. After six more days an agonizing pain in head aroused by a trivial cause; a distressful, restless, but not usually painful head throughout; and finally a sudden snap, and death after twenty-two days' illness.

No autopsy was permitted. The outset of the illness was very peculiar, some of the indications during its course equivocal, one period reassuring and thereby misleading — other periods absorbingly distressing, and the termination unexpectedly sudden.

It was undoubtedly a case of acute infection, and its phenomena can perhaps be explained either on the theory of multiple neuritis or through the action of toxins on the nerve centres, without the existence of localized microscopic lesions. What concerns us here is the question how death was induced.

In the *Revue de Médecine* of February 10, 1891, Dr. Havage, of Paris, reports a case of alleged acute infectious neuritis, in the course of which, and following the abatement of fever, there occurred numbness and prickling, and then extensive paralyses involving not only the limbs but the facial muscles and some of those concerned in articulate speech, and finally the right external muscle of the left eye, inducing strabismus and diplopia. Nevertheless, in a month from the onset of the disease recovery from these paralyses was nearly complete, and the entire restoration to health was not delayed to the end of the second month. Those who cannot share the sanguine belief of Dr. Havage that the case was one of multiple neuritis — a belief that seems scarcely consistent with the general observation of the length of time needed to effect repair and restoration of function in nerve tracts which have been the seat of an inflammatory process — must necessarily regard these formidable paralyses as transitory effects of the toxins of infection.

If, then, such toxins can cause extensive paralyses of peripheral nerves without the aid of inflammation, there is little difficulty in perceiving how they may also, by their depressing, irritating, or paralyzing influence on nerve centres, determine death in various forms, by heart-failure, by asphyxia, and even by pain and shock. Also it is obvious how important must be the influence of family constitution and personal idiosyncrasy in shaping the fatal issue. A single sharp agony sometimes ushers in an attack of localized neuritis; and pain in the head or chest or elsewhere was a notable feature at the critical points in the history of

the family to which I have referred. Was there a correlation between these two facts, or did ptomaines alone cause pain enough to kill? What changes do toxines effect in the nerve elements, to induce now lingering, and again sudden, death?

The questions may not be answered; but the asking serves its purpose of illustrating the obscurities still so often attending the termination, as well as the origin and course, of acute infection.